CASE REPORT

Therapeutic effect of autologous platelet-rich plasma (PRP) on recalcitrant cutaneous ulcers in livedoid vasculopathy

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Livedoid vasculopathy is a chronic disorder diagnosed based on clinical features such as cutaneous ulcers, atrophic, stellate scars with peripheral telangiectasis and hyperpigmentation.[1] Platelet-rich plasma (PRP) therapy has been successfully used in the treatment of various dermatologic conditions including pressure ulcers, diabetic ulcers, and posttraumatic ulcers. The most common PRP therapy is to obtain a sample of blood directly from the patient (autologous).[2,3]

CASE REPORT

A 50-year-old Japanese woman presented with a 20-year history of painful, hyperpigmented, hard, contracted skin with recalcitrant cutaneous ulcers on her lower extremities. She was previously treated with systemic conventional therapies such as prednisolone and warfarin. However, the skin lesions progressed to painful ulcers with eschars and granulation tissue during the last 6 months (Fig 1). Her lower extremities exhibited vascular engorgement in a livedoid pattern over her legs. A biopsy showed dermal fibrosis, intravascular hyalinized material of fibrin-containing thrombi with cell inflammation, and capillary dilatation in the papillary to middle dermis consistent with the clinical diagnosis of livedoid vasculopathy (Fig 2).

Bacterial culture and Gram stain were negative. We obtained 60 mL of peripheral autologous blood, collected into tubes containing acid-citrate-dextrose solution formula anticoagulant. The citrated blood was centrifuged at room temperature for 15 minutes at 1100 rpm. After the plasma layer containing the platelets was collected, the obtained platelet plasma was centrifuged for 15 minutes at 2700 rpm. The supernatant (called "platelet-poor plasma") was removed from the platelet sedimentation after centrifugation, and then the PRP was adjusted at 10-fold concentrations by using 6 mL of platelet-poor plasma. After completion of 3 cycles of autologous PRP treatments at monthly intervals, the lesion showed improvement with atrophic scars and minimal pain (Fig 3). During the PRP therapy, the patient received nonsteroidal anti-inflammatory drug.

Fig 1. Painful ulcers with eschars and granulation tissue on right arch of foot before first platelet-rich plasma therapy.

DISCUSSION

Pathogenesis of livedoid vasculopathy remains unclear and classification is difficult as a result of variable nomenclature and the multifactorial nature of the disease. Idiopathic forms, association with immune complex, and dermal blood vessel occlusion have been described.[4] There is, however,

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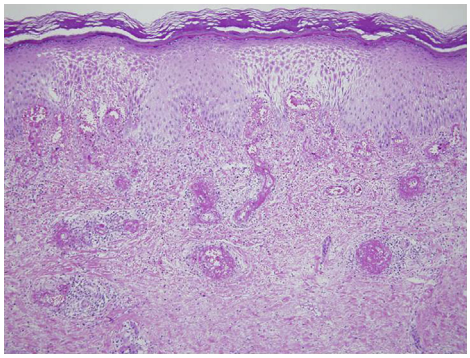

Fig 2. Skin biopsy specimen of the lower leg revealed microvascular thrombi within the vessel wall, erythrocyte extravasation, and inflammatory cell infiltration in the papillary and mid dermis. (Hematoxylin-eosin stain; original magnification: ×100.)

Fig 3. Skin ulcers improved after platelet-rich plasma therapy.

increasing evidence that the condition must be mediated by either coagulation or fibrinolysis disorders.[5-7] Systemic therapeutic recommendations mainly refer to antiplatelet, antithrombotic, anticoagulant, and fibrinolytic medications, which are often unsatisfactory.[1,8,9] We believe this is the first report of successful treatment of recalcitrant cutaneous ulcers in a patient with livedoid vasculopathy by autologous PRP. We propose that autologous PRP therapy could be an appropriate alternative modality when the degree of healing with conventional treatment appears to be unsatisfactory.

PRP consists of a high concentration of platelets that promote wound healing through chemotaxis, cell proliferation, angiogenesis, and tissue remodeling. Platelets are critically important in the wound-healing process. They translocate rapidly to the wound site and adhere to the damaged tissue, initiating a healing reaction that includes the release of various cytokines and growth factors. Topical application of PRP to acute and chronic skin ulcers significantly accelerated the epithelization process, likely through up-regulation of the cell cycle regulatory proteins cyclin A and CDK4.[10] We propose that these mechanisms could accelerate the healing of recalcitrant cutaneous ulcers in livedoid vasculopathy. Additional randomized, controlled clinical trials are required to adequately address the localized treatment effects.

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